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Periodicity in the waxing and waning of Influenza A H1N1: A report from a tertiary care center in Chennai India

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Background: Influenza A H1N1 re emerged in 2009 causing a pandemic and continues to circulate worldwide seasonally. This study was undertaken to characterize Influenza A H 1N1 in a tertiary care center over six years.

Methods & Materials: Throat swabs/nasopharyngeal samples were collected from patients who reported an influenza like illness(ILI)sent in viral transport medium in refrigerated condition to the laboratory. RNA was extracted from the clinical specimens. One step Real Time Reverse Transcriptase Polymerase Chain Reaction was performed (Ambion kit) using specific primers (INF A, universal SwineA, Swine H1 and RNaseP) and the Taqman probe (CDC protocol). Amplification was performed using Real time PCR (ABI 7900HT) system (reverse transcription for 30min at 50^0C and initial activation for 10min at 95^0C followed by 45 cycles of primer annealing and extension at 95^0C for 15sec and 55^0C for 30sec).

Results: A total of 2382 samples were analyzed, n = 480(20.15%) tested positive for novel Influenza A H1N1 and n = 102 (4.28%) tested positive for seasonal influenza A. The positivity rate was 46.08% in 2009, fell from 15.02% (2010), to 1% by 2011; was 22% in 2012 just 1.86% in 2013, 7.096% in 2014 and is currently 44.2% as of Oct 2015. The age of patients ranged from 10 days to >81 years. Influenza Ah1n1 accounted for 25.35% of ILI in children (0-18 years) . The rate of positivity in adults was found to be similar across age groups of 21-60 years (range 19.7%- 21.1%) and declined to 13.5% in greater than 61 years of age. Cough (84.2%) was the predominant symptom, followed by fever (83.75%), breathlessness (55.2%), body ache (54.8%), vomiting (21.4%), diarrhoea (9.58%), 4.4% gave history of travel abroad. Based on the duration of illness the rate of positivity was found to be 78.54% from day one to day 7 of ILI. However 5.62% were positive between day 10 to 30.

Conclusion: There is a cyclical occurrence in the seasonality of Influenza H1N1. Vulnerable groups such as pregnant and lactating mothers may require targeted intervention.

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Clinical features, cytokine profiles and immune response in children with severe hand foot and mouth disease in Vietnam

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Background: Hand, foot and mouth disease (HFMD) is an emerging infection in Asia. Neurological complication and fatality are typically associated with enterovirus A71 (EV-A71). Intravenous immunoglobulin (IVIg) is widely used in the treatment of severe cases, albeit without clinical evidence. This study characterized clinical features, virology, cytokine profiles and immune responses in severe HFMD children receiving IVIg.

Methods & Materials: A prospective study was conducted in the ICU of Children's Hospital 2 (Ho Chi Minh City, Vietnam) from June to September 2012 enrolled HFMD children with clinical indication to have CSF taken. Clinical data, diagnostic throat/rectal swabs, CSF after IVIg, plasma before and after IVIg and at discharge were collected. Plasma cytokines/chemokines and antibodies against common HFMD associated viruses were assessed before and after IVIg and at discharge. Cytokines/chemokines were assessed in CSF.

Results: Thirty patients were enrolled (grade 2b, n=15, grade 3, 13 and grade 4, 2). Twenty-five patients recovered, 2 died and 3 with unknown outcome due to withdrawing. Clinical characteristics improved within 48 hours of admission in grade 2b and after 72 hours in grade 3 patients, coinciding with 1st and 2nd doses of IVIg administration. EV-A71 was detected in swabs of 25 patients and in CSF of 1, and other EVs in swabs of 3. The level of 10 cytokines/chemokines in CSF was comparable between patients receiving one and two IVIg doses, which were also unchanged before and after IVIg in plasma. At discharge, plasma IL-1β, IL-6, IL-10, GM-CSF and IFN-γ were significantly decreased (Figure 1). CSF IL-1β, IL-4, IL-10 and TNF-α were significantly lower as compared to that in plasma after IVIg. In contrast, CSF IL-6 was significantly higher than that in plasma after the 2nd IVIg ($P < 0.05$) (Figure 2). Before IVIg, plasma neutralizing antibody levels against EV-A71 subgenogroup C4 and B5 were high but that against CV-A10/A16/12 were low (Figure 3). These titers increased after IVIg except for that against CV-A12.